



Gut microbiota-derived butyrate mediates the anticolitic effect of indigo supplementation through regulating CD4⁺ T cell differentiation

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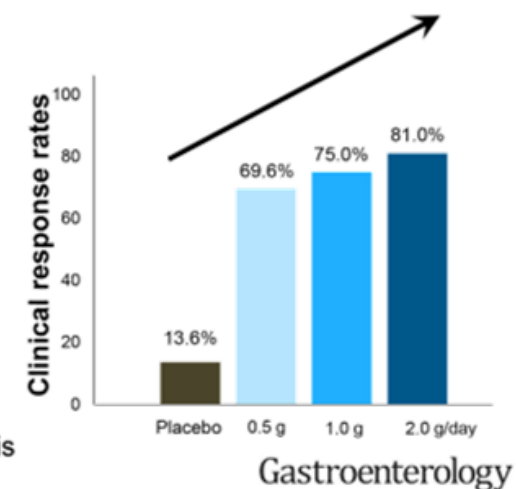
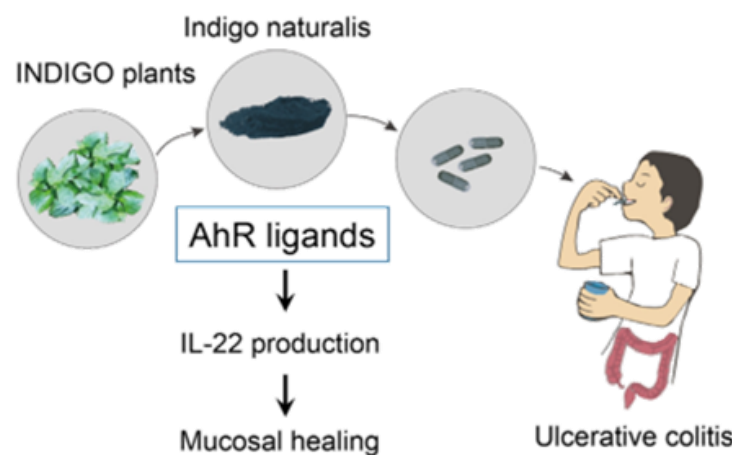
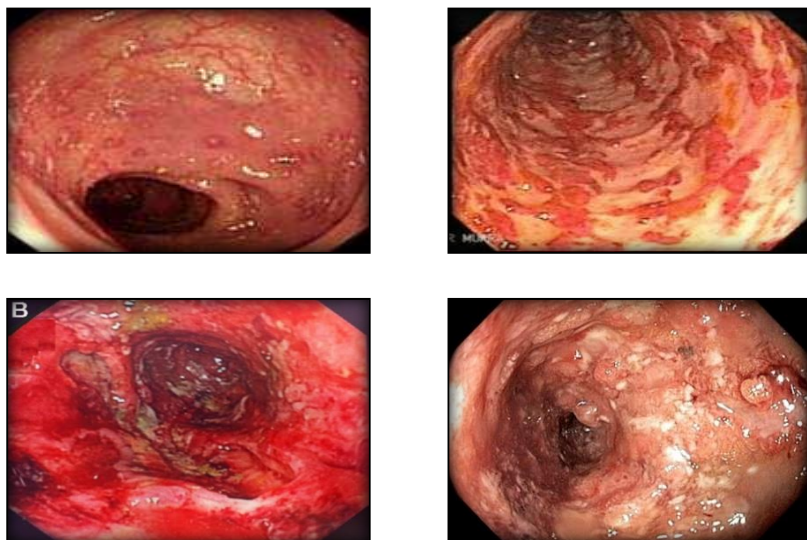
Xing, Yunqi, Muyuan Wang, Yali Yuan, Jiayan Hu, Zhibin Wang, Zhongmei Sun, Mengyu Zheng, Lei Shi, Junxiang Li, and Tangyou Mao. 2025. "Gut Microbiota-Derived Butyrate Mediates the Anticolitic Effect of Indigo Supplementation Through Regulating CD4⁺ T Cell Differentiation." *iMeta* 4: e270040.

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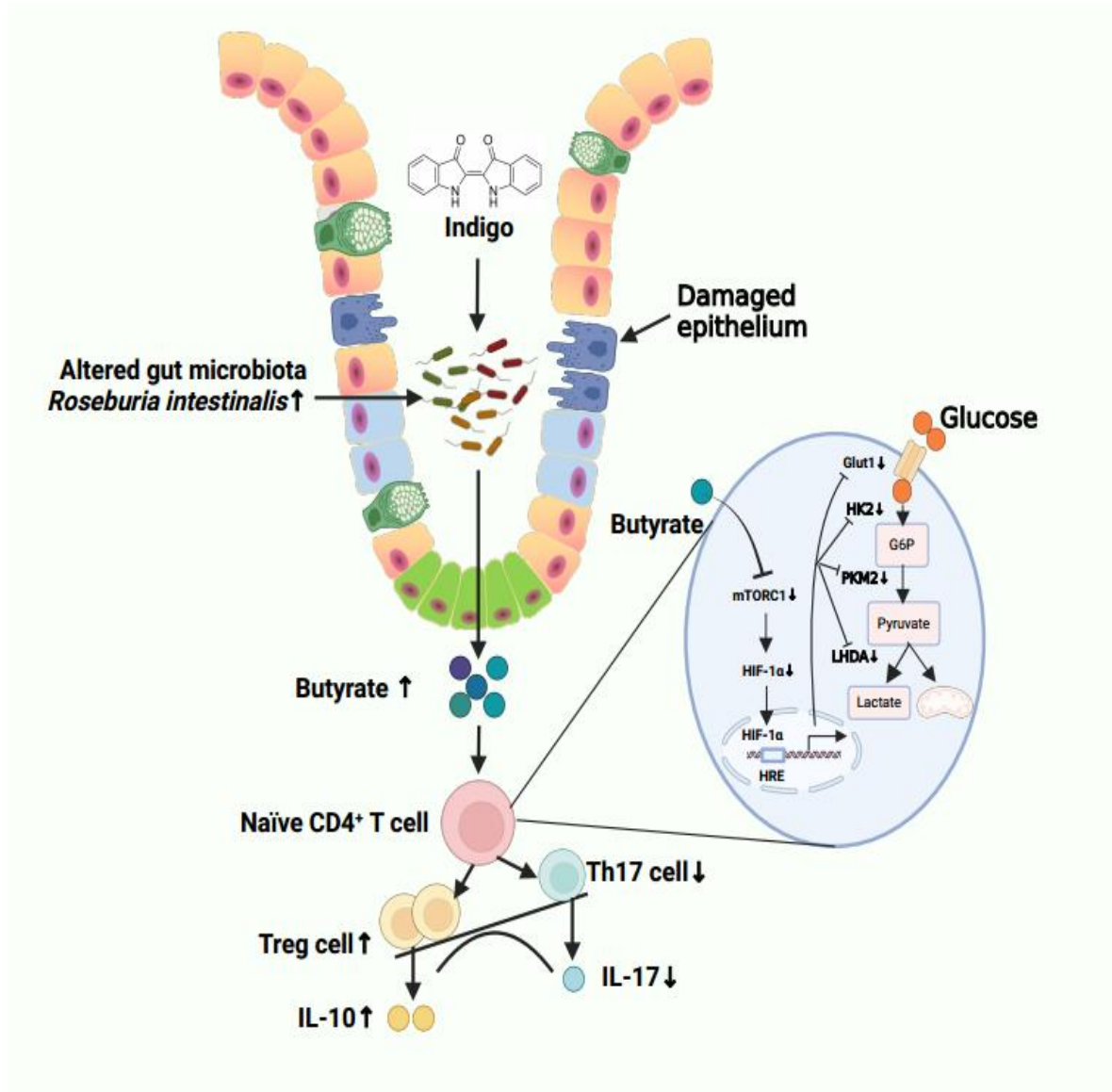
研究背景

- ❑ Ulcerative colitis (UC) is a chronic and recurrent inflammatory intestinal disorder characterized by abnormal mucosal immunity. Current therapies focus on inducing and maintaining disease remission. These approaches can achieve certain therapeutic effects in the short term but have several limitations.
- ❑ Emerging evidence indicates that the gut microbiota and its metabolites are essential for T cell differentiation and activation and maintenance of the Th17/Treg immune balance.
- ❑ Indigo, a pharmacologically active component of indigo naturalis, exhibits multiple anti-inflammatory activities. However, its effects and underlying mechanisms on the complex interface between the intestinal epithelium, microbiota, and mucosal immunity in UC, remain elusive.





亮点

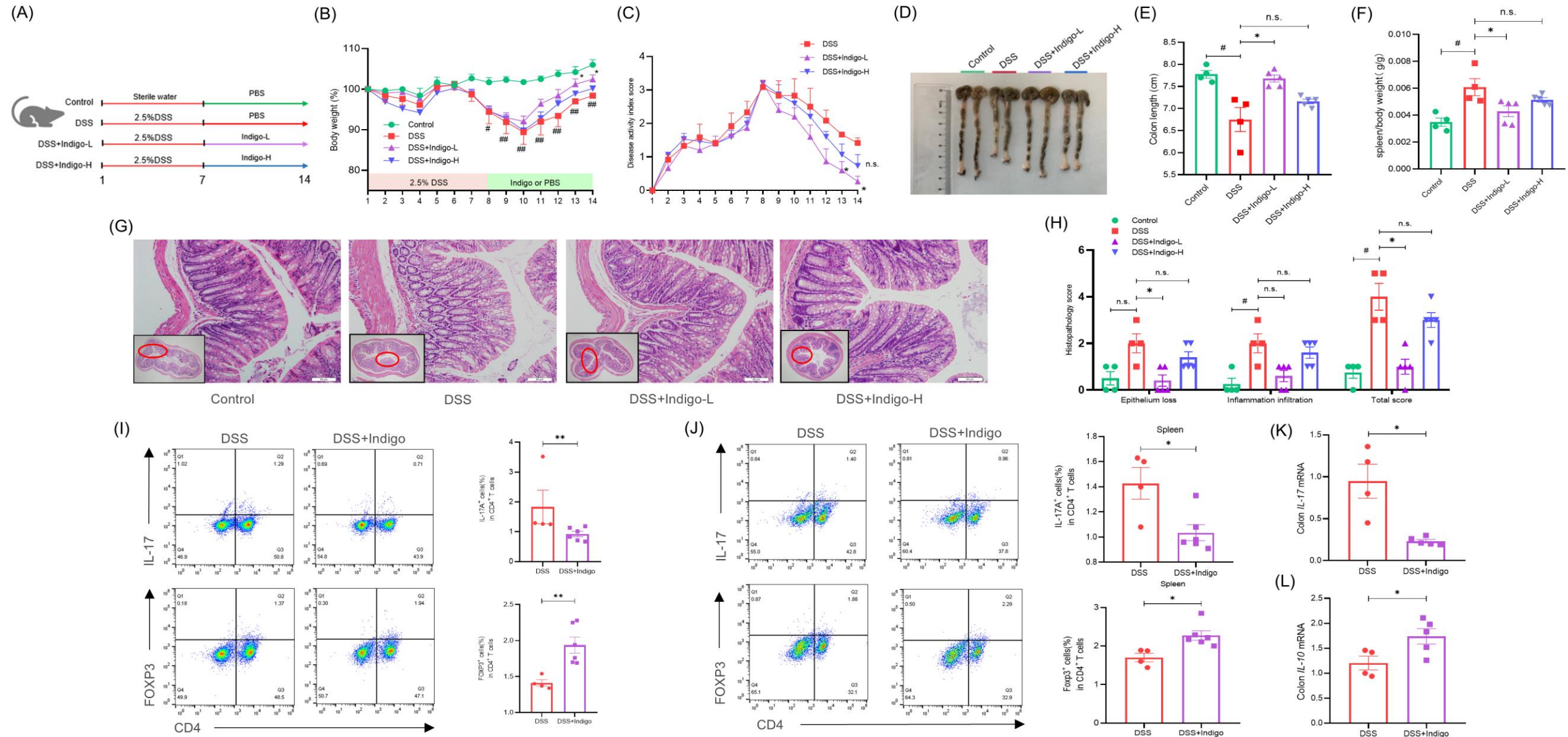


- This study systematically revealed the molecular mechanism by which indigo, an active ingredient in the traditional Chinese medicine, regulates the gut microbiota, especially enriching *R. intestinalis*, and remodels the immune homeostasis of ulcerative colitis. This provides a new strategy for the development of new drugs for ulcerative colitis based on the gut microbiota - immune balance.



结果

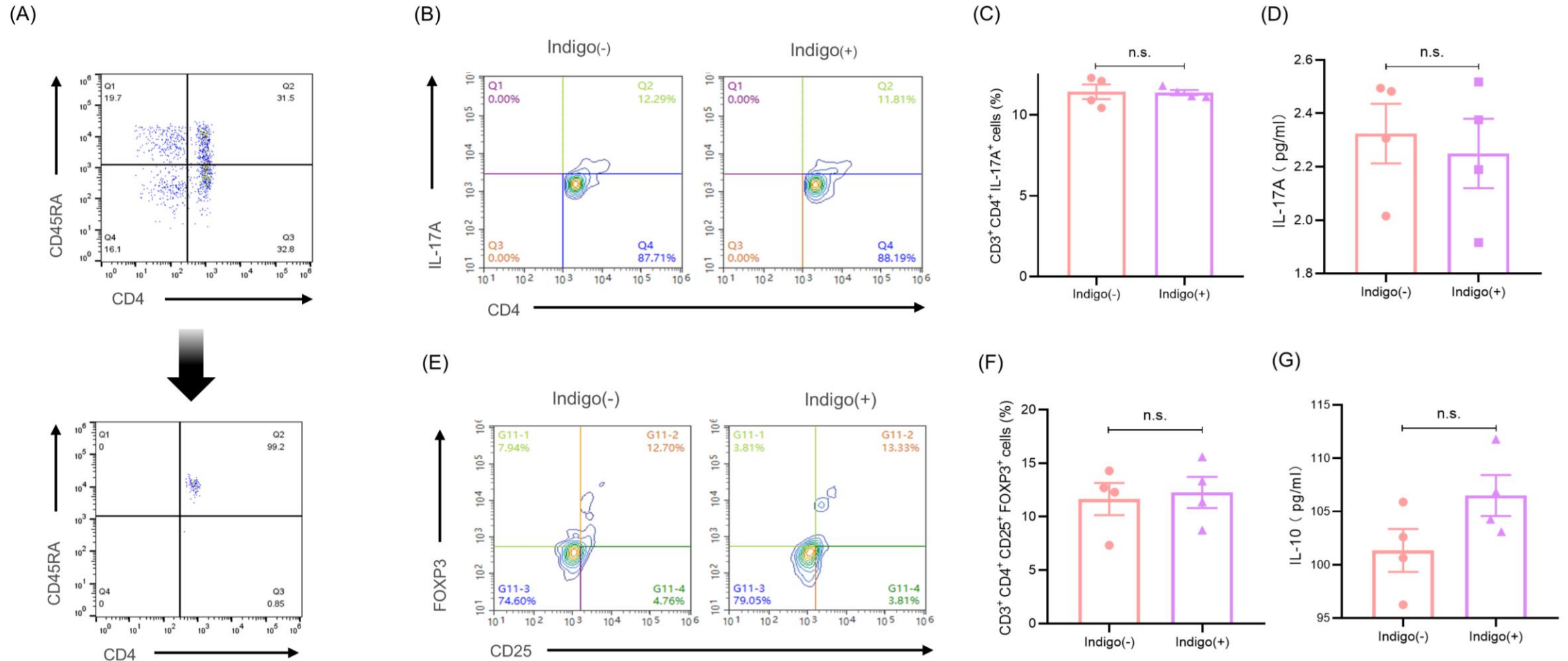
Administration of indigo ameliorated intestinal inflammation and improved Th17/Treg cell balance in colitis mice.





结果

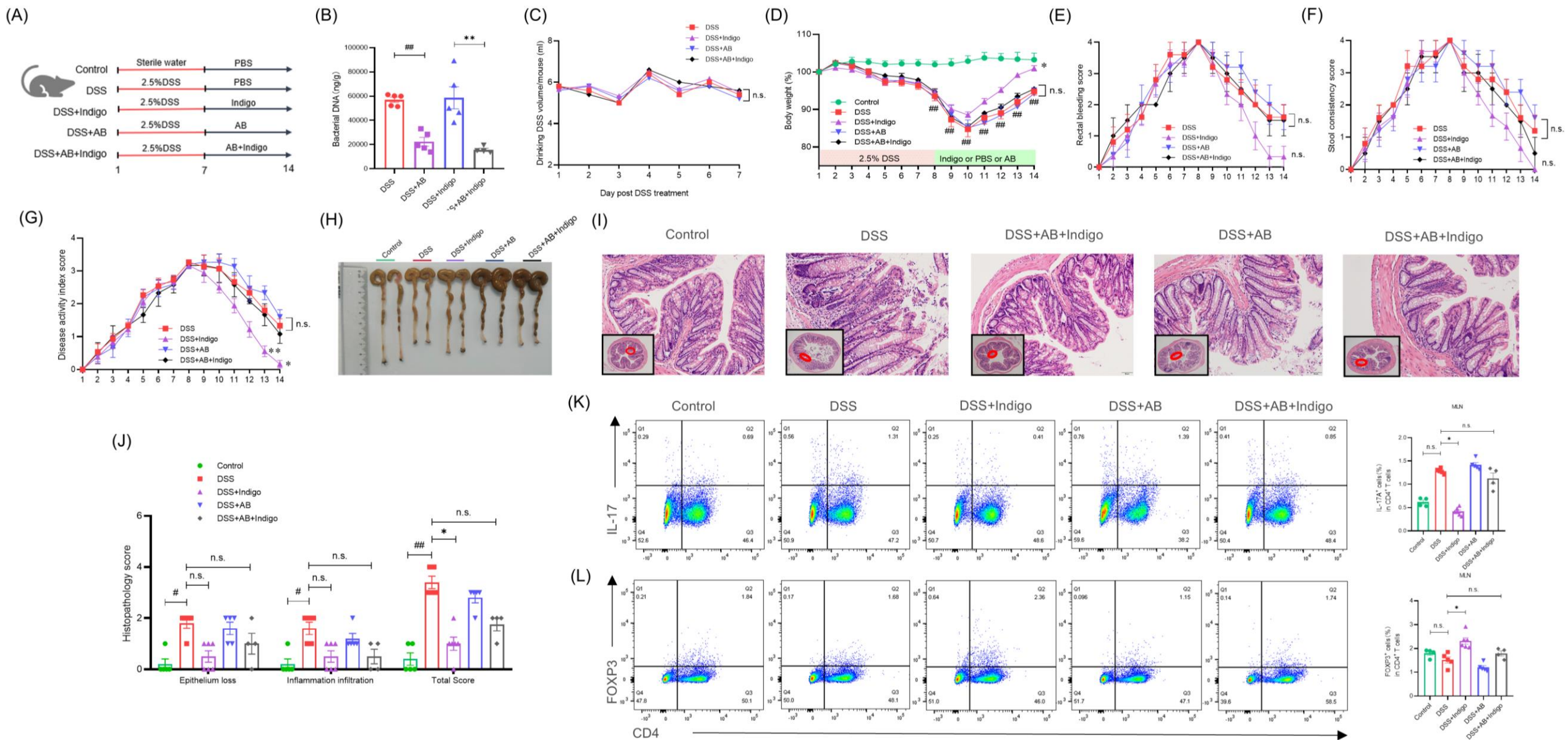
□ Indigo does not directly affect the differentiation of naïve CD4⁺ T cells into Th17/Treg cells, but acts through an unknown indirect pathway.





结果

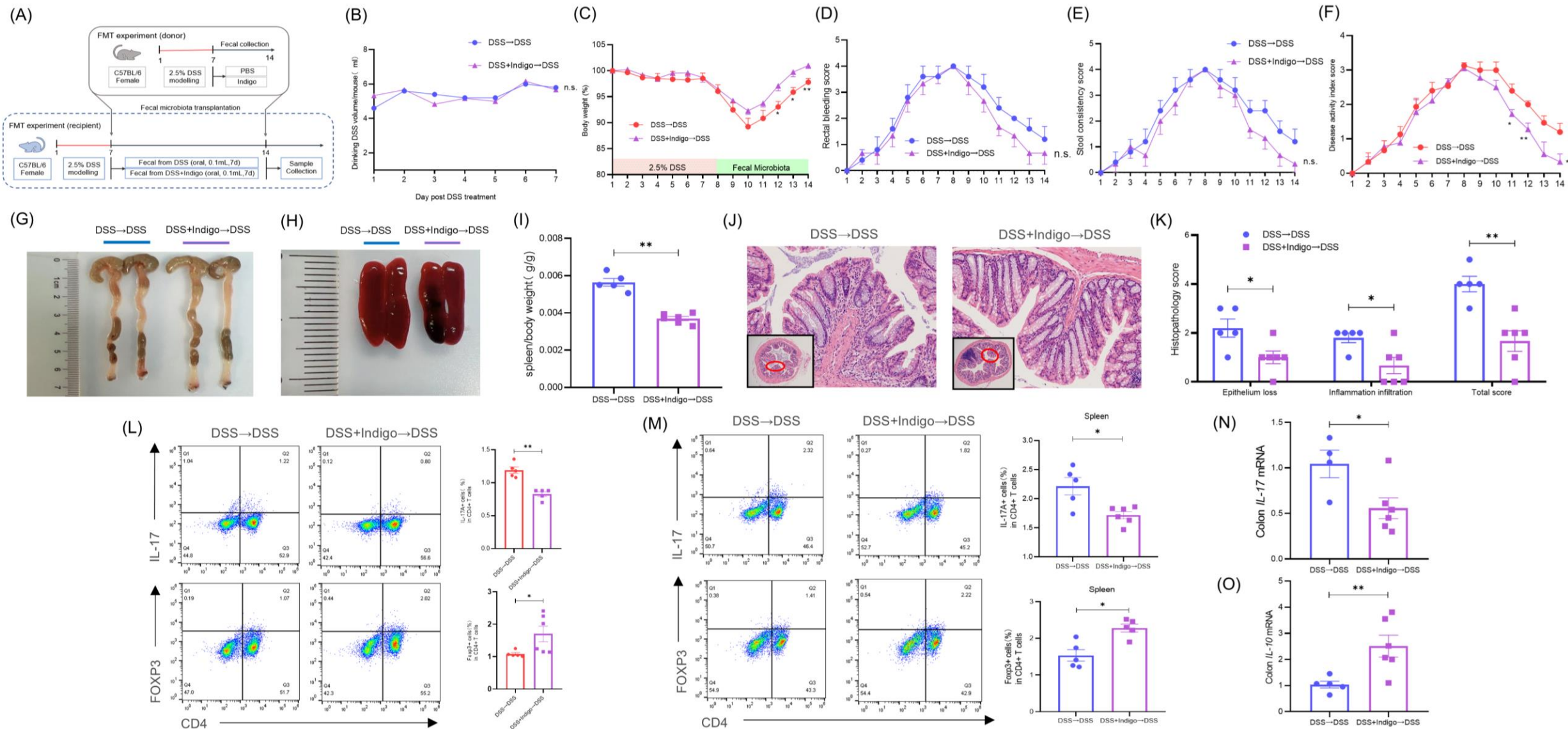
Depletion of the gut microbiota significantly diminished the beneficial effects of indigo on colitis mice.





结果

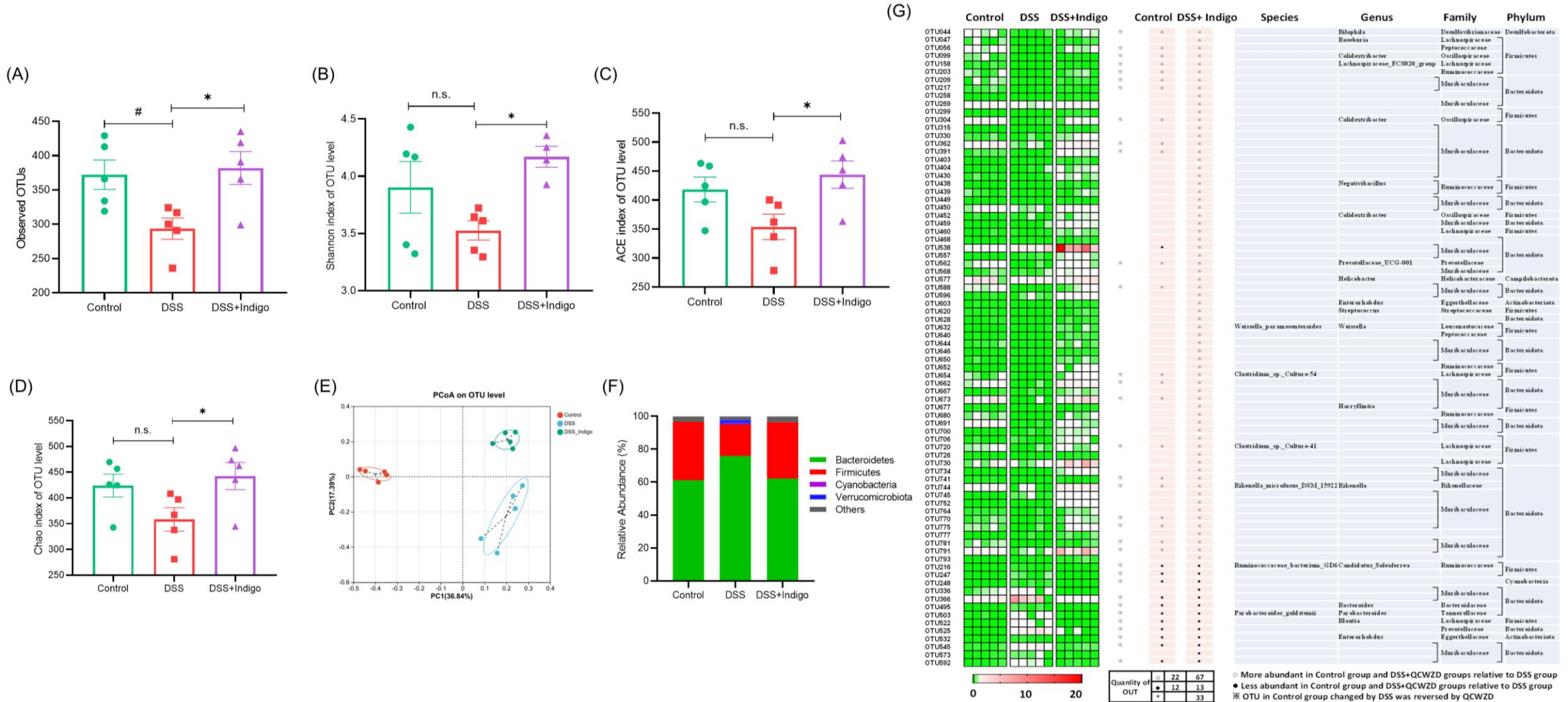
Colitis mice that received microbiota from indigo-treated donors experienced less intestinal inflammation and a reconstructed Th17/Treg balance.





结果

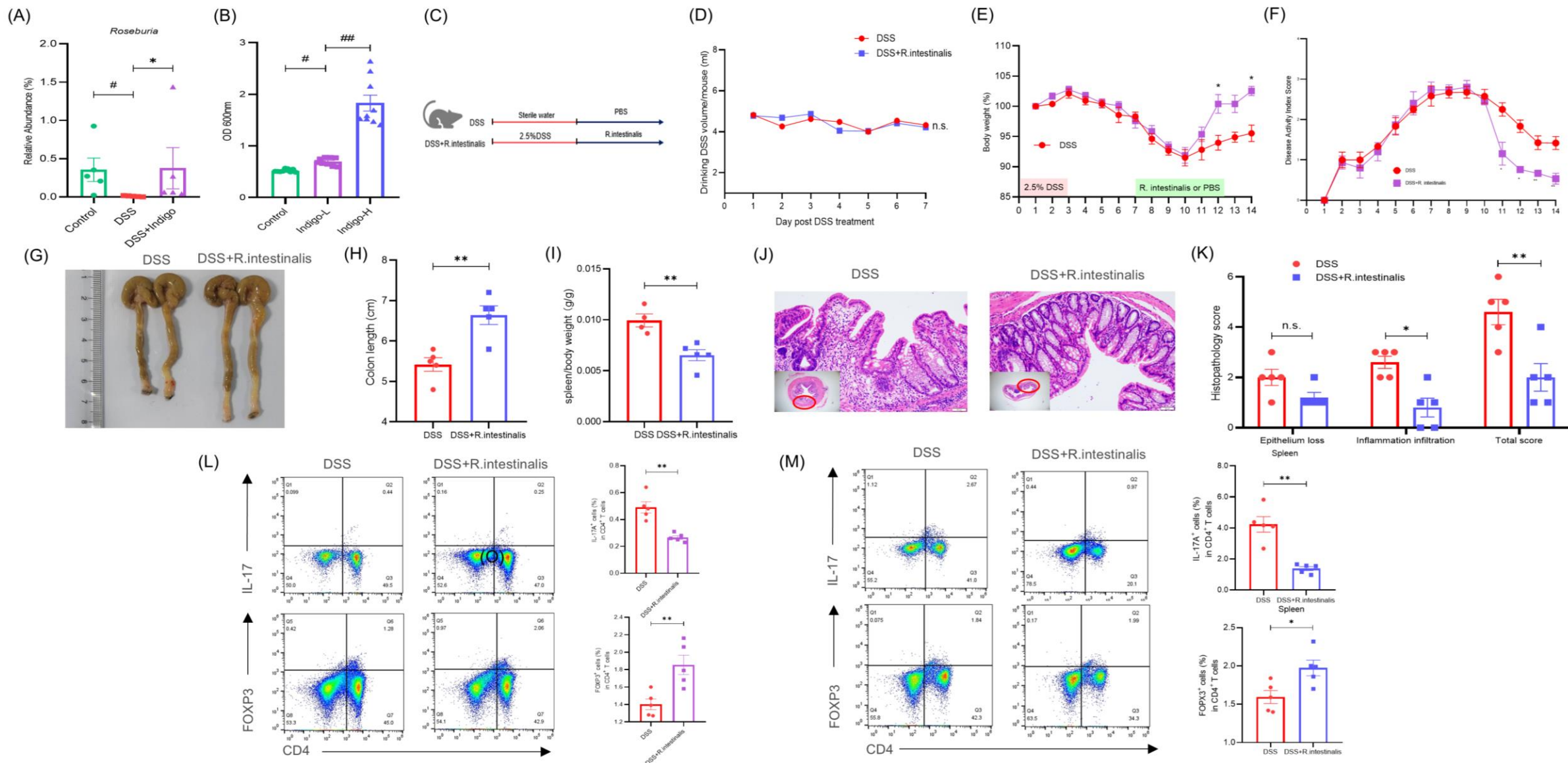
Indigo regulates the gut microbiota of mice, especially by up-regulating the content of *Roseburia* in feces.





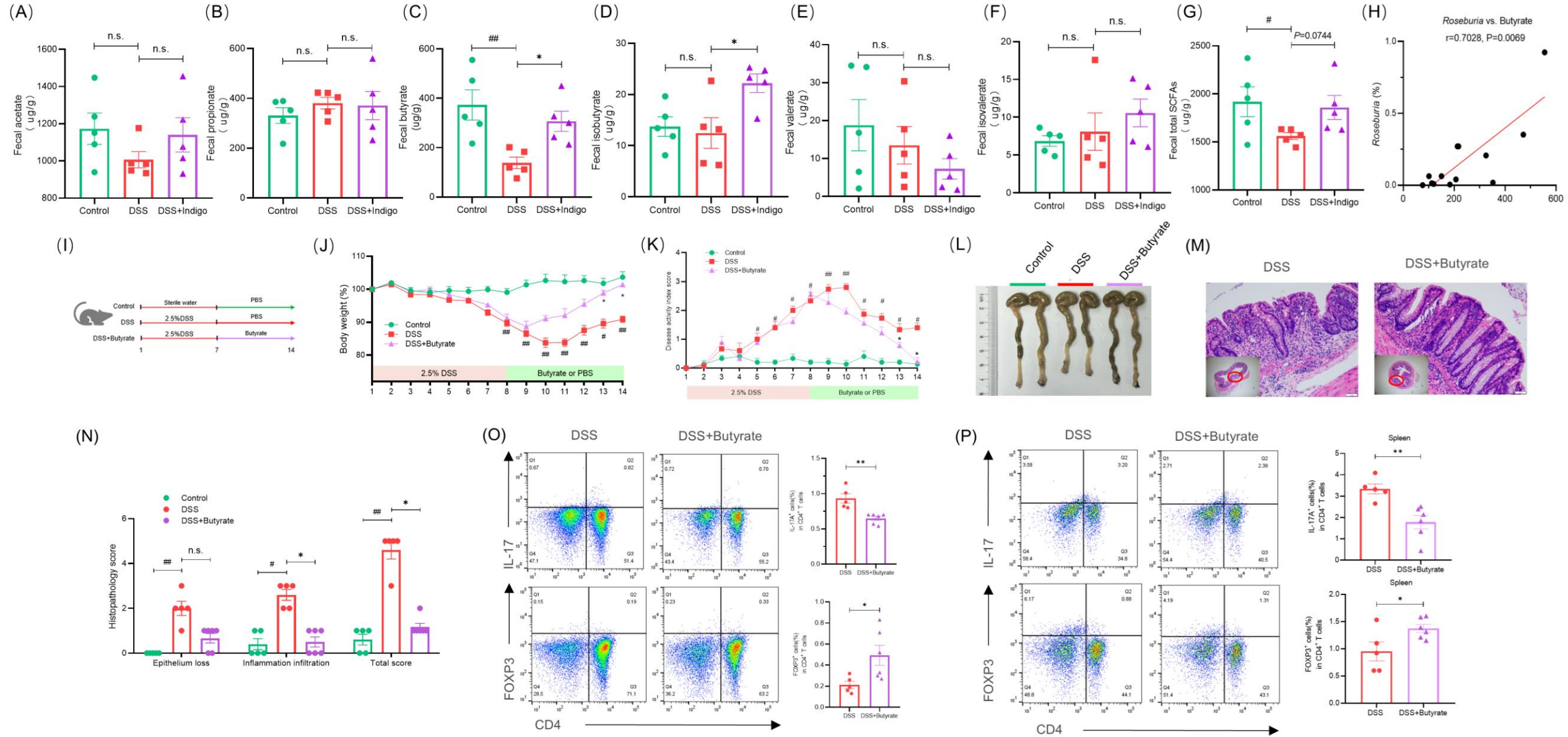
结果

Indigo-induced enrichment of *Roseburia intestinalis* alleviates intestinal inflammation and Th17/Treg balance in mice.



结果

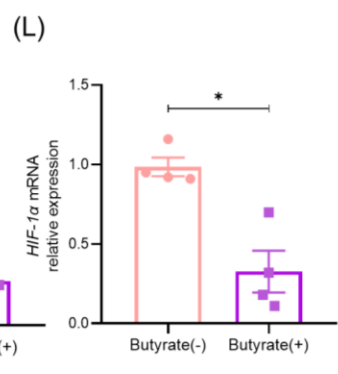
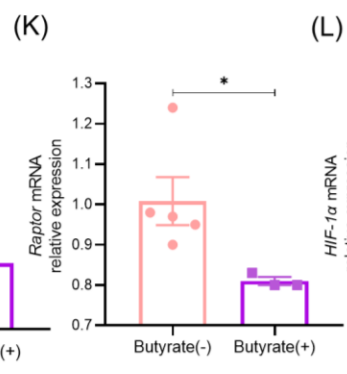
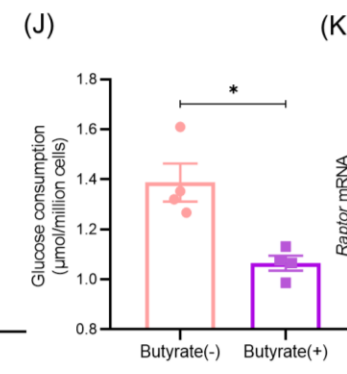
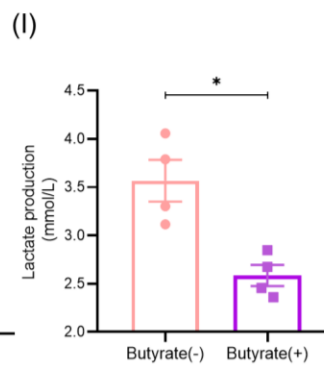
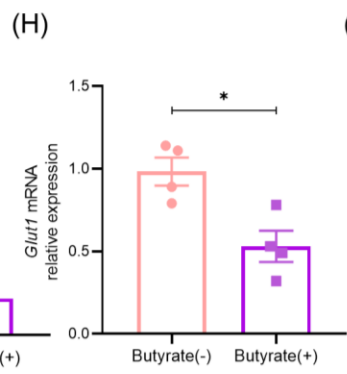
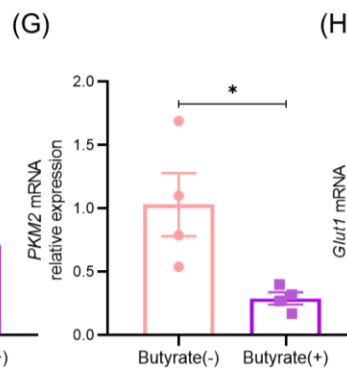
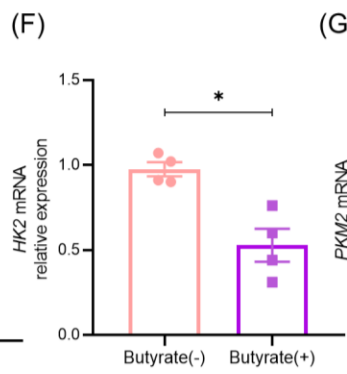
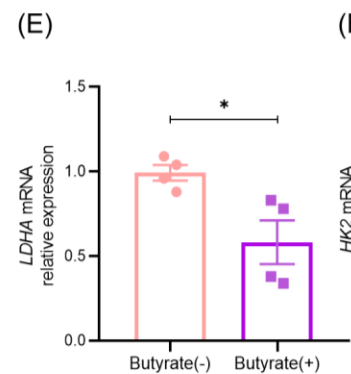
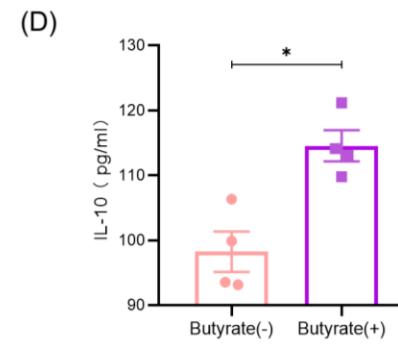
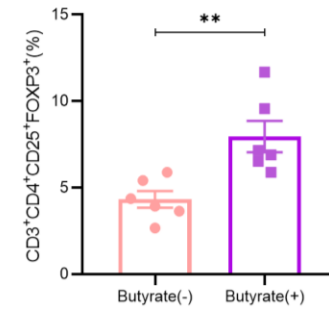
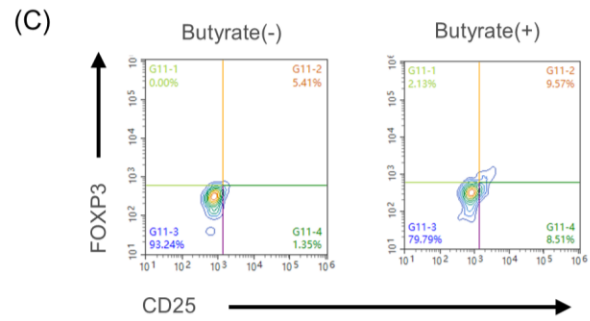
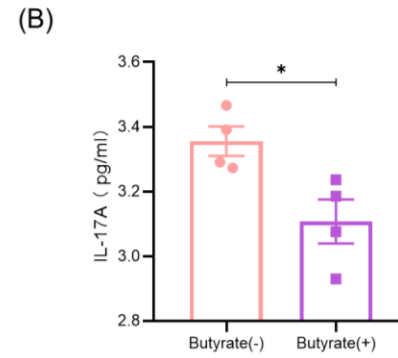
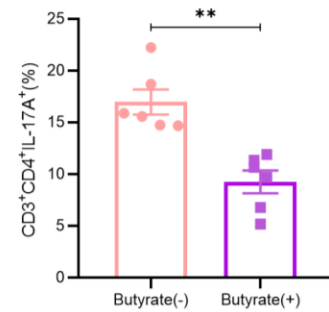
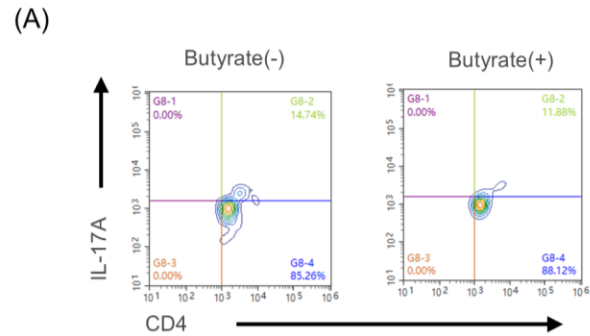
□ Gut microbiota-derived butyrate accelerates intestinal mucosal healing and improve the Th17/Treg balance.





结果

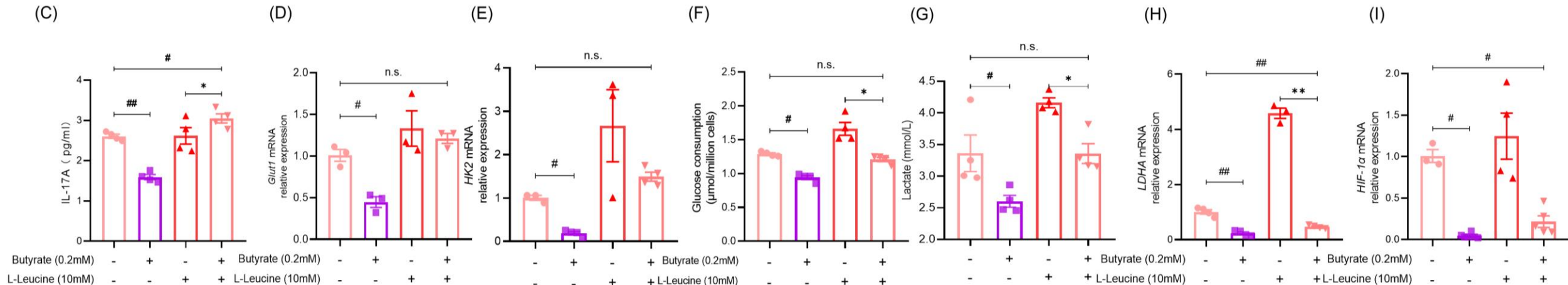
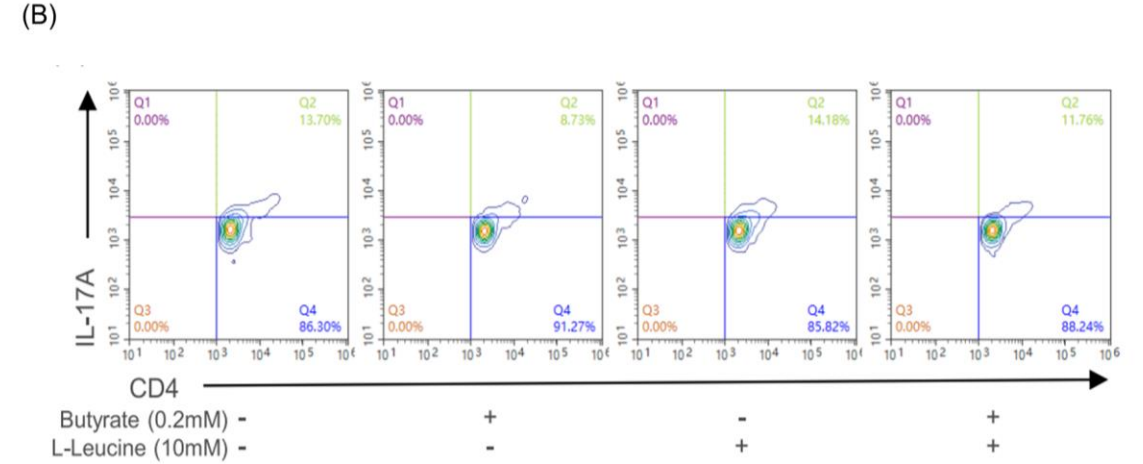
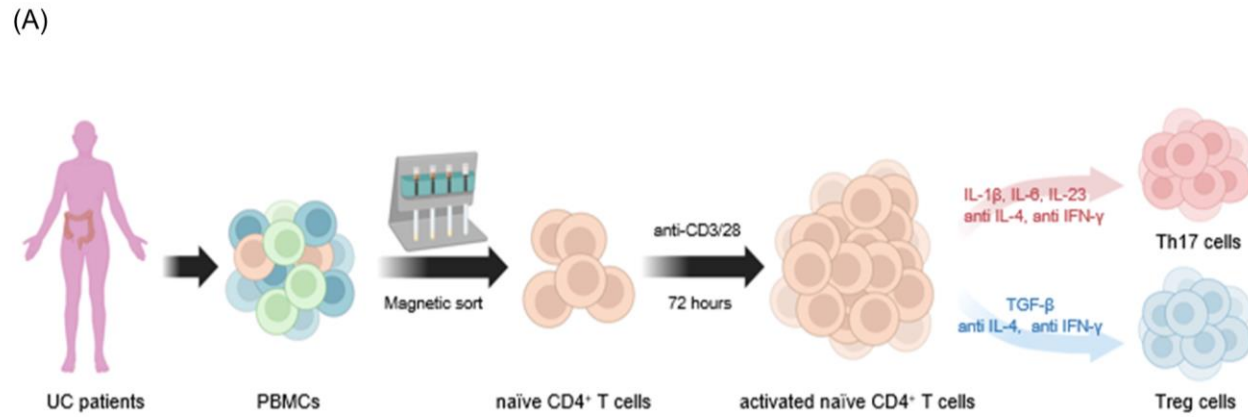
□ Butyrate significantly regulates the differentiation of naïve CD4⁺ T cells *in vitro*.





结果

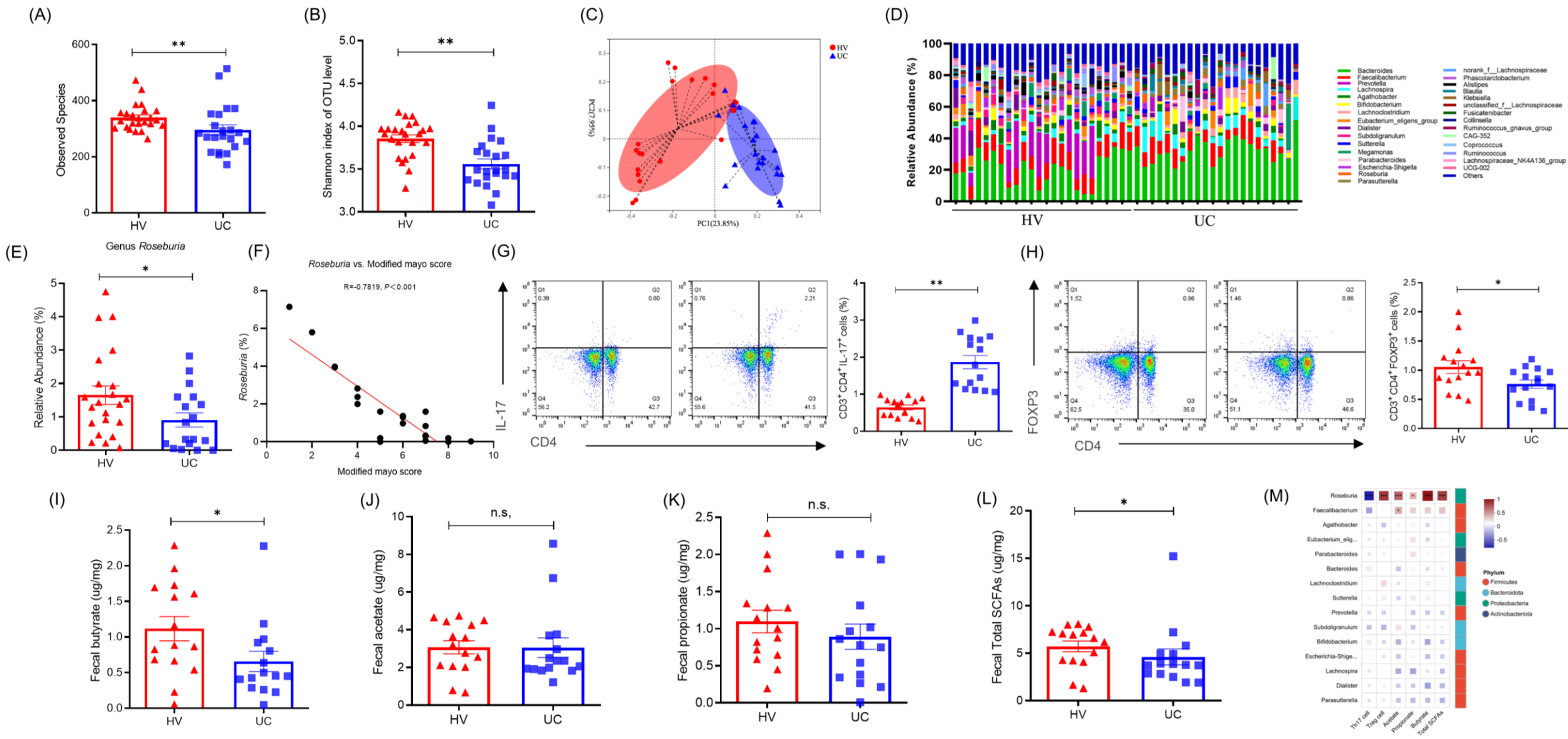
□ The mTORC1/HIF-1 α signal is involved in the differentiation of naïve CD4⁺ T cells induced by butyrate.





结果

Relative abundance of genus *Roseburia* correlate with Th17/Treg cells and fecal butyrate level in UC patients.





总结

- ❑ Indigo alleviates intestinal inflammation in mice with UC by regulating the Th17/Treg immune balance.
- ❑ The gut microbiota, especially *R. intestinalis*, mediates the immune protective effect of indigo against UC.
- ❑ Indigo-enriched *R. intestinalis* metabolite butyrate regulates the differentiation of naïve CD4⁺ T cells through mTORC1/HIF-1 α signal-mediated metabolic reprogramming.
- ❑ UC patients show Obvious Th17/Treg imbalance, gut microbiota dysregulation and decreased butyrate level.




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